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Hyperfibrinogenemia and Increased Stiffness of Plasma Clots in the Active Systemic Lupus Erythematosus

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Abstract Systemic lupus erythematosus (SLE) is an autoimmune disease associated with an increased risk of thrombosis. We hypothesized that inflammation-associated hyperfibrinogenemia can contribute to the prothrombotic phenotype of fibrin clots by changing their mechanical properties. Twenty-eight SLE patients were categorized based on their disease activity scores (SLEDAI) into the groups with inactive (SLEDAI < 4, n = 14) and active (SLEDAI > 4, n = 14) forms of the disease. Clots from individual platelet-free plasma samples were probed using shear rheometry and viscoelastic properties of the fibrin gels were determined as the storage (G') and loss (G'') moduli. A significant increase of G' was revealed in the clots from the plasma of active SLE patients over inactive SLE, which correlated with elevated fibrinogen levels. Clots from the plasma of inactive SLE patients had the elasticity and fibrinogen levels indistinguishable from those in control plasma from healthy subjects. Thus, inflammatory hyperfibrinogenemia in the active SLE form makes fibrin clots stiffer which has been previously shown to be associated with a higher incidence of thrombotic disorders.

Keywords Systemic lupus erythematosus · Fibrin · Shear rheometry

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1 Introduction

In systemic lupus erythematosus (SLE), a multiorgan autoimmune disease, thrombosis makes an important contribution to mortality and long-term morbidity [1]. Although thrombotic complications are relatively common in SLE, mechanisms underlying the SLE-related thrombophilia remain unclear. A network of fibrin fibers is a major component of hemostatic blood clots and obstructive thrombi, which is responsible for their mechanical strength, integrity, and deformability in a highly dynamic environment of flowing blood [2]. Abnormal mechanical properties of fibrin clots have been shown to be associated with a higher risk of thrombosis and thromboembolism [3]. Clot mechanics depend strongly on the variable structure of fibrin that is largely determined by many environmental factors, including a level of fibrinogen, the soluble fibrin precursor in blood [4]. Because fibrinogen is an acute-phase protein that is up-regulated in response to inflammation [5], it is likely to be a pathogenic modulator of the structure and mechanical properties of clots and thrombi. All these provide a rationale for studies on the link between fibrinogen levels in blood, rheological properties of blood clots, and predisposition to thrombosis in SLE. Here we investigated the mechanical features of fibrin clots and their association with fibrinogen levels in SLE patients with respect to the disease activity.

2 Material and Methods

The study was approved by the Ethical Committee of Kazan State Medical University. Twenty-eight consecutive patients who met the revised criteria for SLE of the American College of Rheumatology [6] were enrolled in the study. Twenty-three (82%) of them were females and the mean age of the patients was 37 ± 13 years. The control group comprised 10 age- and